AMENDMENTS TO THE SPECIFICATION

On page 11, please replace the paragraph beginning on line 22 and ending on line 30 with the following amended paragraph:

Figure 3 (SEQ ID NO:208) shows the structure of the *AtFtn2* gene (Panel A) and protein (Panel B). Panel A shows that the open reading frame is terminated by a TAA in-frame stop codon. The diagram depicts introns (thin lines) and exons (black boxes). Sizes are given in bp. The position of the *arc6-1* mutation (C -> T) at position 1141 is marked. The nucleotide sequences flanking the mutation (underlined) show the change of codon 325 (CGA in a wild type plant) into a premature stop (TGA) in *arc6-1*. Panel B shows the putative functional and conserved protein domain, which are depicted as wider black boxes; their numerical positions within the AtFtn2 sequence are also indicated. Black lines above the diagram delineate regions of AtFtn2 conserved among Ftn2 homologues (see Figures 4-6). CT, chloroplast targeting signal.

On page 12, please replace the paragraph beginning on line 24 and ending on line 25 with the following amended paragraph:

Figure 8 (SEQ ID NOS:125-194) shows nucleotide and amino acid sequences of Ftn2 homologs described in Table 3.

On page 39, please replace the paragraph beginning on line 8 and ending on line 29 with the following amended paragraph:

DnaJ domains are characteristic of a family of molecular chaperones. Proteins in this family, from bacterial to human, have three distinct domains: (i) a highly conserved J domain of approximately 70 amino acids, often found near the N-terminus, which mediates interaction of DnaJ (a.k.a., Hsp40) with Hsp70 (DnaK) and regulates the ATPase activity of the latter; (ii) a glycine and phenylalanine (G/F)-rich region of unknown function that may act as a flexible linker; and (iii) a cysteine-rich region (C domain) that contains four CXXCXGXG (SEQ ID NO:207) motifs, and resembles a zinc-finger domain (Ohtsuka K, and Hata M (2000) Int. J. Hyperthermia). Although not originally identified as an *fts* gene, *dnaJ* shares with *fts* genes the property that its inactivation leads to a filamentous phenotype (Paciorek J, Kardys K, Lobacz B, and Wolska KI (1997) Acta Microbiol. Pol. 46:7-17). Cheetham and Caplan (Cheetham ME, and Caplan AJ (1998) Cell Stress Chaperones 3:28-36) classified DnaJ/Hsp40 homologs into three groups: type I have all three of these domains; type II have only the J and G/F domains; and type III, like Ftn2,

have only a J domain. DnaK proteins are highly versatile chaperones that assist a large variety of processes (Bukau B (1999 ed.) Molecular Chaperones and Folding Catalysts-Regulation, Cellular Function and Mechanisms, Hardwood, Amsterdam; Bukau B, and Horwich AL (1998) Cell 92:351-366; Cai Y, and Wolk CP (1997) J. Bacteriol. 179:258-266; Fink A (1999) Physiological Rev. 79:425-449; Gething MJ (1997) Nature 388:329-331; Hartl FU (1996) Nature 381:571-579), from folding of newly synthesized proteins to facilitation of proteolytic degradation of unstable proteins (Laufen T, Mayer MP, and Heiter P (1995) Sci. USA 96:5452-5457). This functional diversity requires that DnaK proteins associate promiscuously with misfolded proteins or selectively with folded substrates, including with regulatory proteins of low abundance.

Please replace the Sequence Listing attached herewith for the Sequence Listing filed February 20, 2004.